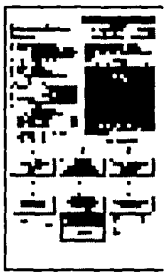
Title: **JP61155335A2: IMMUNOMODULATOR**

Country: **JP** Japan
Kind: **A**



Inventor(s): **WATANABE MASAHIRO**
NAKAJIMA TSUNETAKA
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Applicant/Assignee: **GREEN CROSS CORP:THE**

Issued/Filed Dates: **July 15, 1986** / Dec. 28, 1984

Application Number: **JP1984000276758**

IPC Class: **A61K 39/395;**

Priority Number(s): Dec. 28, 1984 **JP1984000276758**

Abstract: **Purpose**: An immunomodulator useful for rheumatic diseases, collagen diseases, etc., having excellent immunomodulating action, and low toxicity, comprising at least one selected from blocked Fc fragment, blocked Fab fragment, blocked L chain and blocked H chain of human IgG as an active ingredient.
Constitution: An immunomodulator comprising at least one selected from blocked Fe fragment, blocked Fab fragment, blocked L chain, and blocked H chain of human IgG as an active ingredient. The blocked Fc fragment and the blocked Fab fragment are obtained by scissoring the disulfide bond of Fc fragment and Fab fragment of human IgG, and blocking the formed SH group of each fragment. The immunomodulator acts in an adjuvant way or an immunosuppressive way depending upon immune response condition of host. This drug can be administered in the form of injection or oral drug. It has low toxicity and excellent immunomodulating action, so it is useful for immunodeficiency diseases such as rheumatic diseases, collagen diseases, etc.
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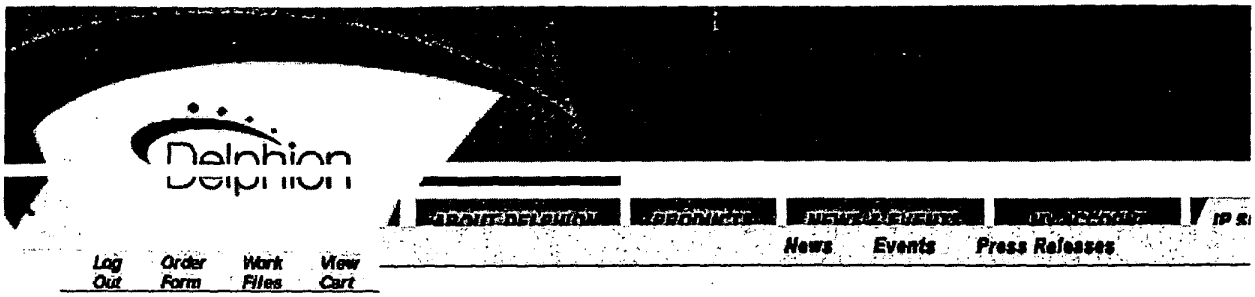

The Delphion Integrated View

Other Views:
INPADOC | Derwent...

Title: **JP2104532A2: DRUG COMPOSITION SHOWING IMMUNE MODULATION ACTIVITY**

▶ Want to see a more descriptive title highlighting what's new about this invention?

Country: **JP** Japan
Kind: **A**

Inventor(s): **LUGARO GIUSEPPE**
DI SAN SECONDO VITTORIO ROSSO

No Image

Applicant/Assignee:
CONSIGLIO NAZI RICERCHE
OSPEDALE MAGGIORE POLIOLINICO
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Issued/Filed Dates: **April 17, 1990** / May 23, 1988

Application Number: **JP1988000125613**

IPC Class: **A61K 37/02;** A61K 37/02; A61K 37/02; A61K 35/14;

▶ Interested in classification by use rather than just by description?

Priority Number(s): May 28, 1987 **IT1987000020701**

Abstract:

Purpose: To obtian a medicinal composition which contains a low-molecular peptide fraction extracted from seminal plasma and manifests immune modulation activity, particularly immunosuppression activity.
Constitution: This medicinal composition contains a peptide fraction with a molecular weight of about 1,000 extracted from seminal plasma as an active ingredient. The single dose of this compound is 1-100μg/kg in parenteral administration and 10-1,000μg/kg in oral administration. This composition is effective for symptoms caused by insufficiency of suppresser cell activity (for example, allergic diseases due to IgE), symptoms caused by insufficiency of suppresser cell activity relating to abnormal activity or inadequate activity of helper cells (such as rheumatoid arthritis), transplantation of organs and tissues, some kinds of infectious diseases showing immune-attacking properties, for example, chronic hepatitis or congenital immunodeficiency syndrome.

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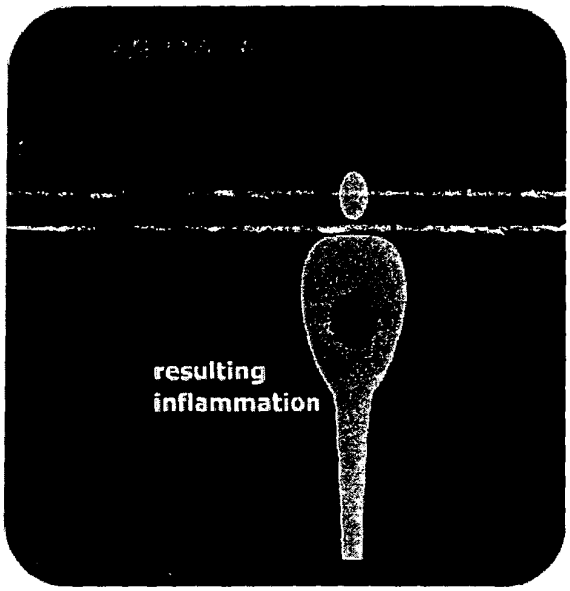
▶ S a clear and precise summary f th wh l pat nt, in und rstandable terms.

TNF-α Modulation



Inflammation

Inflammation can be caused by a number of inflammatory agents, e.g. bacteria, viruses, injury, etc. These agents promote inflammation at a cellular level by binding to the cell surface. Once a cell becomes inflamed it releases pro-inflammatory proteins called cytokines. These messenger proteins now cause neighboring cells to become inflamed, initiating an inflammatory cascade.

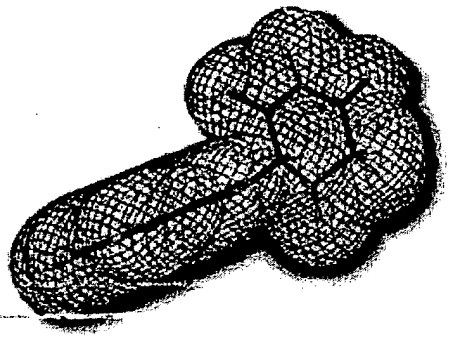
When an immunomodulatory drug is administered, it blocks the inflammatory signal initiated at the cell surface. This results in a down-regulation of the inflammatory cascade.

Celgene has made substantial progress in its research on Tumor Necrosis Factor Alpha (TNF-α) as a target for new pharmaceuticals. TNF-α is a protein manufactured by cells of the immune system. At normal levels, the protein is essential for effective immune function. Overproduction of TNF as a result of age, genetic and other influences, however, contributes to the pathology of numerous diseases.

Researchers are attempting to suppress TNF-α overproduction in two ways: By inactivating

circulating levels of TNF-α through large-molecule antibodies, or soluble receptors. By limiting synthesis or release of TNF-α from the immune system, i.e., by attacking TNF-α overproduction at its source.

Celgene has identified this route as being accessible through its two classes of small molecule, orally available agents: SelCIDs™ and IMiDs™. These agents use different biochemical mechanisms to target underlying TNF-α overproduction without effecting general immune system function.



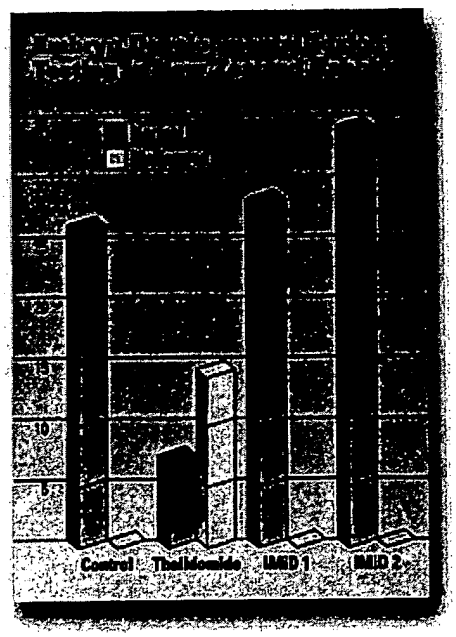
IMiDs™- More Potent

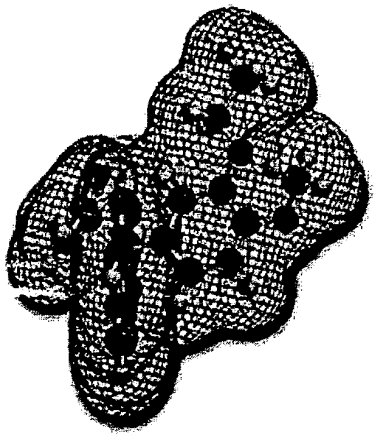
Based on the biological activity and structure of THALOMID® (thalidomide), Celgene has designed and synthesized a number of novel structural analogues called Immunomodulatory Drugs, or IMiDs™.

The IMiDs™ compounds developed by Celgene's research organization have demonstrated an ability to inhibit TNF-α production in vitro with a potency more than 10,000 times greater than THALOMID® (thalidomide).

IMiDs™- Pre-Clinical Toxicity

In preliminary non-primate tests, IMiDs™ do not appear to be teratogenic (cause birth defects), in cases where the same dosage of thalidomide led to fetal deformities.





SelCIDs™

Celgene scientists have developed a new range of compounds called Selective Cytokine Inhibitory Drugs, or SelCIDs™. SelCIDs™ have been determined to have an inhibitory effect on phosphodiesterase type 4 enzyme (PDE-4). The inhibition of PDE-4 may result in decreased Tumor Necrosis Factor Alpha (TNF-α) production.

High levels of tumor necrosis factor-alpha (TNF-α), a key mediator of inflammation produced by monocytes, can cause several different inflammatory disorders in humans. Fortunately, production of TNF-α can be blocked by increasing intracellular levels of cyclic adenosine monophosphate (cAMP). When a β2 agonist binds to its cell surface receptor (R), stimulatory G-protein (Gs) becomes activated, which in turn activates adenylyl cyclase (AC) to produce cAMP. Normally, cAMP is converted to AMP through the action of phosphodiesterase type 4 (PDE4). However, in the presence of PDE4 inhibitors, cAMP levels remain high, causing activation of protein kinase A (PKA). PKA activity prevents transcription factors such as NF-κB from promoting transcription of the TNF-α gene, thus resulting in a blockade of TNF-α production.

PDE4 Inhibitors Block TNF-α Production